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MRI of Focal Fibrocartilaginous Dysplasia

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Summary: Focal fibrocartilaginous dysplasia (FFCD) is an uncommon, benign condition associated with unilateral tibia vara in young children. The clinical, pathologic, plain film, and magnetic resonance imaging (MRI) findings of FFCD were reviewed in two children. MRI findings were virtually identical in both patients and correlated well with the plain film and pathologic findings. We believe that FFCD has a typical MRI appearance. However, FFCD also has characteristic plain film findings, and when these are present, MRI is indicated for only an atypical clinical presentation. **Key Words:** Focal fibrocartilaginous dysplasia—Tibia vara.

Focal fibrocartilaginous dysplasia (FFCD) is an uncommon, benign condition associated with unilateral tibia vara in young children. Since its description in 1985 (1), a total of 16 cases has been reported (1–5,7). The etiology of FFCD is unknown; however, Bell et al. hypothesized that it may be due to abnormal development of the mesenchymal anlage at the insertion of the pes anserinus in the tibial metaphysis (1). The appearance of this condition on computed tomography (CT) was described by Herman et al. in 1990 (3). We present the clinical, pathologic, plain film, and magnetic resonance imaging (MRI) findings in two children with FFCD.

CASE REPORTS

Case 1

A 7-month-old boy was first seen with bowing of his right leg. This was initially treated with bracing. A plain film (Fig. 1A) obtained at 18 months of age showed tibia vara with a well-defined, cortical lucency and distal sclerosis involving the medial aspect of the proximal tibial diametaphysis. The patient's deformity persisted, and at 24 months of age, MRI (Fig. 1B, C) was performed to define the bone lesion further and to exclude an adjacent soft-tissue mass. Two months later, the patient underwent open biopsy of the tibial lesion with tibial and fibular osteotomies. Biopsy revealed dense tendinous connective tissue with ossification and reactive bone consistent with the diagnosis of FFCD. Postoperatively, the patient has done well, and the tibial deformity has not recurred.

Case 2

A 12-month-old boy was first seen with a history of left tibial bowing since birth. A radiograph (Fig. 2A) showed findings consistent with FFCD. MRI (Fig. 2B) was performed, and 2 months later, the patient underwent biopsy of the tibial lesion with tibial and fibular osteotomies. Biopsy revealed a sheet of dense fibroconnective tissue intermingled with reactive immature woven bone with bony cartilaginous transition resembling callus, consistent with FFCD. A follow-up radiograph showed healing osteotomies with resolution of the tibial deformity (Fig. 2C).

MRI FINDINGS

MRI examinations were performed on a 1.5-T Magnetom scanner (Seimens Medical Systems, Iselin, NJ, U.S.A.) and included T1-weighted [short repetition time (TR)/short echo time (TE)] and T2-weighted (long TR/long TE) sequences. The MRI findings were virtually identical in both patients. Areas corresponding to the cortical lucency on plain films were low signal on both short TR/short TE and long TR/long TE images. Areas corresponding to the distal sclerosis were low signal on short TR/short TE images and intermediate signal on long TR/long TE images. No soft-tissue mass was present in either patient.

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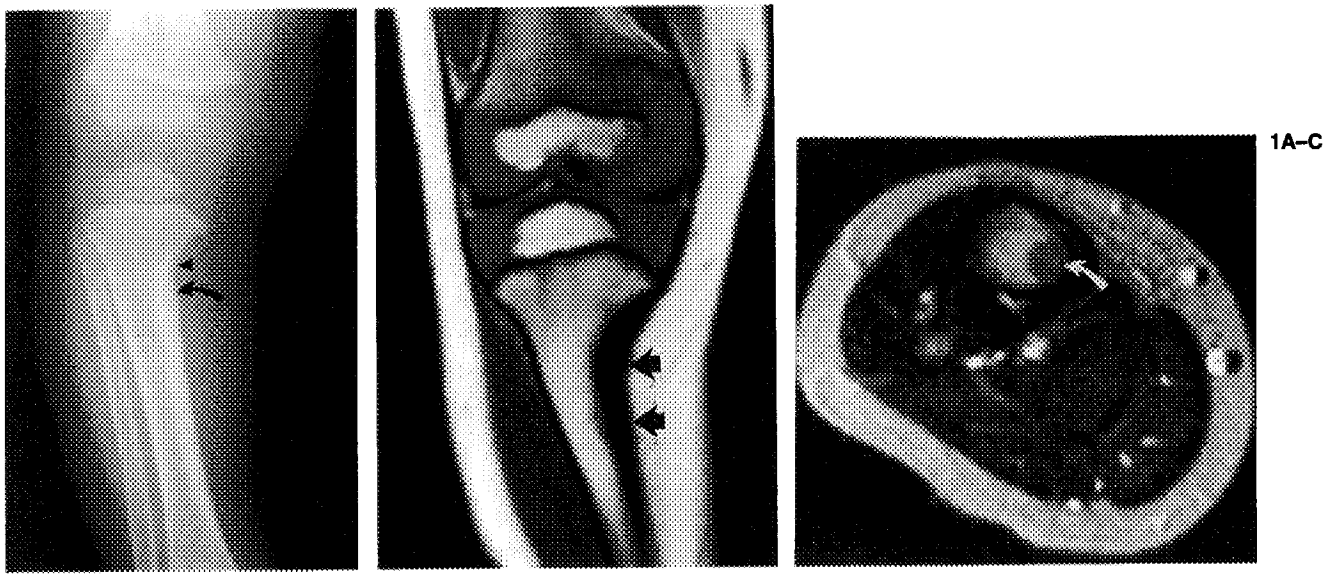

FIG. 1. A: Radiograph shows tibia vara with cortical lucency (arrowhead) and distal sclerosis (curved arrow) involving the medial diametaphyseal region of the tibia. **B:** Coronal short TR/short TE MRI shows tibia vara with low signal (arrows) in areas corresponding to lucency and sclerosis on plain film. **C:** Axial long TR/long TE image shows predominantly intermediate signal (curved arrow) in area of cortical sclerosis on plain film.

DISCUSSION

FFCD presents as a nontraumatic and painless unilateral bowleg deformity with the plain film findings of tibia vara, a well-defined cortical lucency on the medial aspect of the tibial metaphysis, and cortical sclerosis distal to the area of lucency (1–5,7). Radiographically and clinically, FFCD must be distinguished from other causes of tibia vara, such as Blount disease, deformity due to growth-plate closure from trauma or infection, and late-onset tibia vara (6).

In Blount disease, which also occurs in young children, the proximal tibial epiphysis is wedge-shaped, the proximal tibial physis is irregular, and the medial metaphysis of the tibia is beaked (6). In postinfectious or posttraumatic tibia vara, the deformity is centered at the growth plate where a bony bridge causes partial closure, and there are no preceding changes in the shape of the epiphysis or metaphysis (6). Late-onset tibia vara occurs in obese children 6–14 years of age; the epiphyses are wedge-shaped because of medial flattening, and the growth plates are irregular in thickness (6). In contrast, the proximal tibial epiphysis is normal in FFCD, and the tibia vara is centered at the cortical lucency located in the proximal tibial diametaphysis, a short distance distal to a normal tibial growth plate. The cortical lucency and sclerosis of FFCD may suggest other lesions such as eosinophilic granuloma, chondromyxoid fibroma, osteoid osteoma, or osteomyelitis; however, these lesions usually produce soft-tissue masses and are not associated with tibia vara (3).

The treatment of FFCD is controversial. In the initial description by Bell et al., two children underwent osteotomies, and one child was treated with a brace; all three children had good outcomes (1). Since then, another 13 patients have been described in the literature, and of these, eight treated without an osteotomy and four treated with osteotomies have done well (2–5,7). The one remaining patient developed a peroneal nerve palsy and had a persistent valgus deformity after osteotomy (2). When considering surgical intervention, it should be noted some patients had early progression of their tibial deformity, only to have it eventually resolve without treatment (2).

The two patients in this report had clinical, plain film, and pathologic findings of FFCD. Both of these patients had unilateral bowleg deformity and no history of pain or trauma. Like those previously reported (1–5,7), our patients were seen before 3 years of age and had characteristic plain film findings of FFCD. Pathology obtained in each case was consistent with the "dense hypocellular tissue resembling fibrocartilage in some areas and tendon in others" described by Bell et al. (1).

The MRI findings of FFCD are consistent with the CT findings of an elliptical cortical bony defect without a discrete soft-tissue mass (3) and correlate well with the plain films. The cortical lucency seen on plain films was low signal on short TR/short TE and long TR/long TE images, consistent with the dense fibroconnective tissue seen on pathologic examination. The more distal areas of sclerosis were low signal on short TR/short TE images and intermediate signal on long TR/long TE images, consis-

2A–C

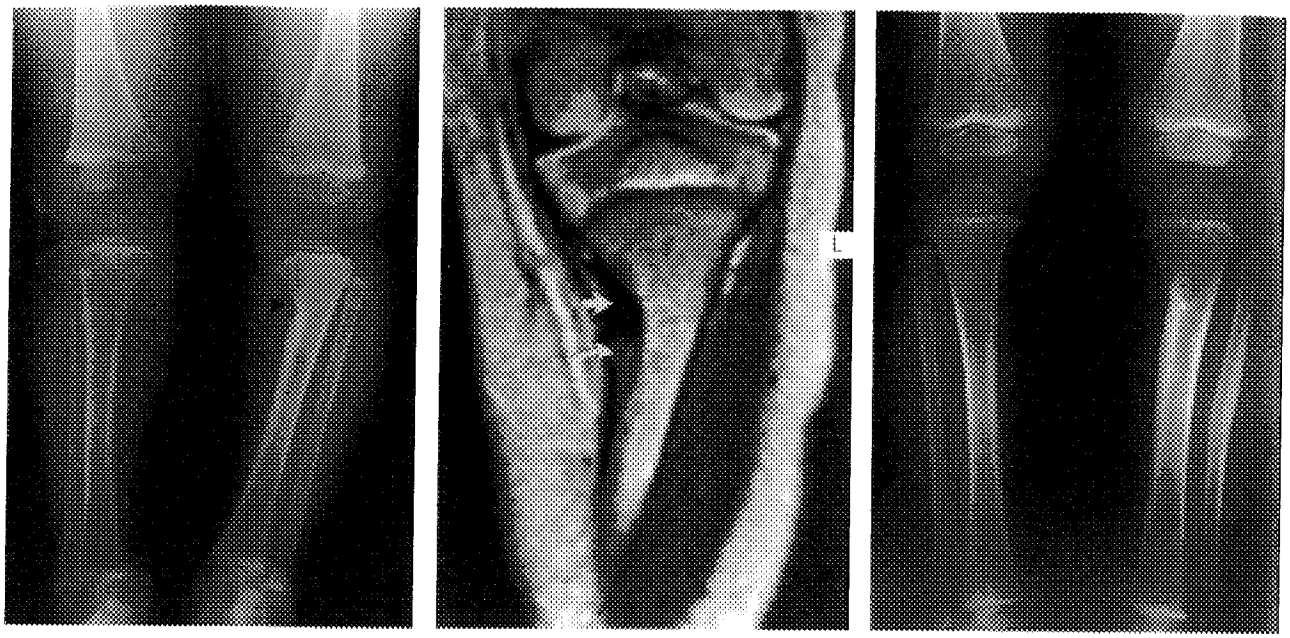

FIG. 2. A: Radiograph shows typical bony findings of focal fibrocartilaginous dysplasia. Note that the cortical lucency (arrowhead) is not contiguous with the growth plate. **B:** Coronal long TR/long TE image shows low signal (arrow) in the area corresponding to radiographic lucency and intermediate signal (curved arrow) in the area corresponding to radiographic sclerosis. **C:** Radiograph showing healing osteotomies and resolution of the tibial deformity.

tent with the reactive bone and callus seen pathologically.

Based on these findings, we believe that FFCD has a typical MRI appearance. However, FFCD also has characteristic plain film findings, and when these are present, we believe that MRI is indicated only as a problem-solving examination in the presence of an atypical clinical presentation.

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